

STIC-ILL

*RC 681 A1 C5*

**From:** Qian, Celine  
**Sent:** Wednesday, February 25, 2004 7:04 PM  
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Please make copy of the following articles. Thank you.

Percutaneous electromechanical mapping demonstrates efficacy of pVGL1 (VEGF2) in an animal model of chronic myocardial ischemia.

AU Vale, Peter R. [Reprint author]; Tkebuchava, Tengis [Reprint author];  
Milliken, Charles E. [Reprint author]; Chen, Donghui [Reprint author];  
Symes, James F. [Reprint author]; Isner, Jeffrey M. [Reprint author]

CS St Elizabeth's Med Ctr, Boston, MA, USA

SO Circulation, (Nov. 2, 1999) Vol. 100, No. 18 SUPPL., pp. I.22. print. *#109*  
Meeting Info.: 72nd Scientific Sessions of the American Heart Association.  
Atlanta, Georgia, USA. November 7-10, 1999.

*Celine Qian*  
Art Unit 1636  
Rensem 2A89  
571-272-0777

# WEST Search History

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<input type="checkbox"/>	L2	VEGF-2 or VEGF-C or VEGF2 or VEGFC or VEGF 2 or VEGF C	834
<input type="checkbox"/>	L1	pVGL1	2

END OF SEARCH HISTORY

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NEWS 16 DEC 19 CROPU no longer updated; subscriber discount no longer available  
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NEWS 22 FEB 05 German (DE) application and patent publication number format changes  
  
NEWS EXPRESS DECEMBER 28 CURRENT WINDOWS VERSION IS V7.00, CURRENT  
MACINTOSH VERSION IS V6.0b(ENG) AND V6.0b(JP),  
AND CURRENT DISCOVER FILE IS DATED 23 SEPTEMBER 2003  
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=> s pVGI.1  
L1 2 PVGI.1

=> dup rem l1  
PROCESSING COMPLETED FOR L1  
L2 2 DUP REM L1 (0 DUPLICATES REMOVED)

=> d bib abs 1-  
YOU HAVE REQUESTED DATA FROM 2 ANSWERS - CONTINUE? Y(N);y

L2 ANSWER 1 OF 2 CAPLUS COPYRIGHT 2004 ACS on STN  
AN 2002:122826 CAPLUS  
DN 136:178364  
TI Vascular endothelial growth factor 2 nucleic acids, polypeptides and polypeptide fragments for use in treating various disease states  
IN Coleman, Timothy A.  
PA Human Genome Sciences, Inc., USA  
SO PCT Int. Appl., 241 pp.  
CODEN: PIXXD2  
DT Patent  
LA English  
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2002011769	A1	20020214	WO 2001-US24658	20010803
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
AU 2001084734	A5	20020218	AU 2001-84734	20010803
EP 1313512	A1	20030528	EP 2001-963814	20010803
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
US 2003215921	A1	20031120	US 2001-921143	20010803
NZ 518077	A	20031128	NZ 2001-518077	20010803
PRAI US 2000-223276P	P	20000804		
WO 2001-US24658	W	20010803		
AB Disclosed are human VEGF-2 polypeptides, bio. active, diagnostically or therapeutically useful fragments, analogs, or derivs. thereof, and DNA (RNA) encoding such VEGF-2 polypeptides. Also provided are procedures for producing such polypeptides by recombinant techniques and antibodies and antagonists against such polypeptides. Such polypeptides and polynucleotides may be used therapeutically for stimulating wound healing and for vascular tissue repair. Also provided are methods of using the antibodies and antagonists to inhibit tumor angiogenesis and thus tumor growth, inflammation, diabetic retinopathy, rheumatoid arthritis, and psoriasis.				
RE.CNT 7	THERE ARE 7 CITED REFERENCES AVAILABLE FOR THIS RECORD			
ALL CITATIONS AVAILABLE IN THE RE FORMAT				

L2 ANSWER 2 OF 2 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN  
AN 2000:30458 BIOSIS  
DN PREV200000030458  
TI Percutaneous electromechanical mapping demonstrates efficacy of \*\*\*pVGI\*\*\* . \*\*\*1\*\*\* (VEGF2) in an animal model of chronic myocardial ischemia.  
AU Vale, Peter R. [Reprint author]; Tkebuchava, Tengis [Reprint author]; Milliken, Charles E. [Reprint author]; Chen, Donghui [Reprint author]; Symes, James F. [Reprint author]; Isner, Jeffrey M. [Reprint author]  
CS St Elizabeth's Med Ctr, Boston, MA, USA  
SO Circulation. (Nov. 2, 1999) Vol. 100, No. 18 SUPPL., pp. I.22. print  
Meeting Info.: 72nd Scientific Sessions of the American Heart Association. Atlanta, Georgia, USA. November 7-10, 1999.  
CODEN: CIRCAZ. ISSN: 0009-7322.  
DT Conference; (Meeting)  
Conference, Abstract, (Meeting Abstract)  
LA English  
ED Entered STN: 13 Jan 2000  
Last Updated on STN: 31 Dec 2001

=> s VEGF2 or VEGF 2 or VEGFC or VEGF C  
L3 1125 VEGF2 OR VEGF 2 OR VEGFC OR VEGF C

=> s l3 and expression vector  
L4 11 L3 AND EXPRESSION VECTOR

=> dup rem l4l  
ENTER L# LIST OR (END):l4  
'L4l' IS NOT VALID. VALID FILE NAMES ARE 'BIOSIS, EMBASE, CAPLUS'  
You have entered a file name of duplicates to keep that is not referenced by any of the L#s specified for this DUPLICATE command. The file names of duplicates that can be kept are listed above. Please enter one of these file names.

=> dup rem l4  
PROCESSING COMPLETED FOR L4  
L5 9 DUP REM L4 (2 DUPLICATES REMOVED)

=> s l5 not l1  
L6 8 L5 NOT L1

=> d bib abs 1-

YOU HAVE REQUESTED DATA FROM 8 ANSWERS - CONTINUE? Y/(N):y

L6 ANSWER 1 OF 8 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS  
INC. on STN

AN 2002:441586 BIOSIS

DN PREV200200441586

TI Modulation of host gene expression by the constitutively active G  
protein-coupled receptor of Kaposi's sarcoma-associated herpesvirus.

AU Polson, Andrew G.; Wang, David; DeRisi, Joseph; Ganem, Don [Reprint  
author]

CS Department of Microbiology and Immunology, 513 Parnassus Avenue, Box  
0414,

San Francisco, CA, 94143, USA

ganem@cgl.ucsf.edu

SO Cancer Research, (August 1, 2002) Vol. 62, No. 15, pp. 4525-4530. print.  
CODEN: CNREA8. ISSN: 0008-5472.

DT Article

LA English

ED Entered STN: 21 Aug 2002

Last Updated on STN: 21 Aug 2002

AB Kaposi's sarcoma-associated herpes virus (KSHV) infects B cells and  
microvascular endothelium, and is linked to both lymphoid and endothelial  
neoplasms. KSHV encodes a G protein-coupled receptor (v-GPCR) that can  
bind several CC and CXC chemokines but is able to signal in the absence of  
known ligands. This signaling can transform cultured fibroblasts, promote  
angiogenesis in vitro and in vivo, and activate the mitogen-activated  
protein kinase, c-Jun-NH2-terminal kinase, and p38 pathways. To assess  
the potential impact of v-GPCR signaling on host cell biology we have  
examined cellular gene expression in v-GPCR-transfected cells using DNA  
microarrays. v-GPCR expression up-regulated numerous cellular transcripts  
in both BJAB B cells and SLK endothelial cells, but with a remarkable  
degree of cell-type specificity. Among the most highly regulated genes in  
endothelial cells were the cytokines interleukin 6 and GROalpha; several  
genes affecting endothelial/vascular growth and remodeling were also  
induced, including plasminogen, thrombospondin, the urokinase-type  
plasminogen activator receptor, and to a modest extent vascular  
endothelial growth factor C. By contrast, the most highly regulated genes  
in B cells were the CC chemokines macrophage inflammatory protein 1alpha  
and macrophage inflammatory protein 1beta. No genes other than members of  
the dual-specificity phosphatase family were induced in both cell lines.  
The results indicate that the effects of KSHV GPCR expression in these two  
target cell types differ considerably and suggest that signaling by this  
molecule may make different contributions to the pathogenesis of  
KSHV-related endothelial and lymphoproliferative lesions.

L6 ANSWER 2 OF 8 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS  
INC. on STN

AN 2002:275961 BIOSIS

DN PREV200200275961

TI NogaTM left ventricular mapping to assess percutaneous catheter-based gene  
transfer of vascular endothelial growth factor 2 ( \*\*\*VEGF\*\*\* - \*\*\*2\*\*\*  
) in a placebo-controlled, double-blind trial of patients with chronic  
myocardial ischemia.

AU Vale, Peter Richard [Reprint author]; Milliken, Charles E. [Reprint  
author]; Fortuin, David; Symes, James F.; Schatz, Richard A.; Losordo,  
Douglas W.

CS St Elizabeth's Med Ctr of Boston, Boston, MA, USA

SO Circulation, (October 23, 2001) Vol. 104, No. 17 Supplement, pp. II.664.  
print.

Meeting Info.: Scientific Sessions 2001 of the American Heart Association.  
Anaheim, California, USA. November 11-14, 2001. American Heart  
Association.

CODEN: CIRCAZ. ISSN: 0009-7322.

DT Conference; (Meeting)

Conference; Abstract; (Meeting Abstract)

LA English

ED Entered STN: 8 May 2002

Last Updated on STN: 8 May 2002

L6 ANSWER 3 OF 8 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS  
INC. on STN

AN 2002:263364 BIOSIS

DN PREV200200263364

TI Inhibition of endothelial cell surface expression of vascular endothelial  
growth factor-2 using an intrabody strategy.

AU Wheeler, Yurong Y. [Reprint author]; Kute, Timothy E. [Reprint author];  
Willingham, Mark C. [Reprint author]; Chen, Si-Yi; Sane, David C.

CS Wake Forest Univ Sch of Med, Winston-Salem, NC, USA

SO Circulation, (October 23, 2001) Vol. 104, No. 17 Supplement, pp. II.68.  
print.

Meeting Info.: Scientific Sessions 2001 of the American Heart Association.  
Anaheim, California, USA. November 11-14, 2001. American Heart  
Association.

CODEN: CIRCAZ. ISSN: 0009-7322.

DT Conference; (Meeting)

Conference; Abstract; (Meeting Abstract)

LA English

ED Entered STN: 1 May 2002

Last Updated on STN: 1 May 2002

L6 ANSWER 4 OF 8 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS  
INC. on STN

AN 2001:322890 BIOSIS

DN PREV200100322890

TI Human cutaneous fatty acid-binding protein induces metastasis by  
up-regulating the expression of vascular endothelial growth factor gene in  
rat Rama 37 model cells.

AU Jing, Chun; Beesley, Carol; Foster, Christopher S.; Chen, Haijuan;  
Rudland, Philip S.; West, David C.; Fujii, Hiroshi; Smith, Paul H.; Ke,  
Youqiang [Reprint author]

CS Molecular Pathology Laboratory, Department of Pathology, University of  
Liverpool, Liverpool, L69 3BX, UK

SO Cancer Research, (June 1, 2001) Vol. 61, No. 11, pp. 4357-4364. print.  
CODEN: CNREA8. ISSN: 0008-5472.

DT Article

LA English

ED Entered STN: 4 Jul 2001

Last Updated on STN: 19 Feb 2002

AB Human cutaneous fatty acid-binding protein (C-FABP) gene is capable of  
inducing the metastatic phenotype when overexpressed in nonmetastatic rat  
Rama 37 cells. However, the mechanism of how it induces metastasis is not  
clear. Northern and slot blot analyses revealed that expression of the  
endogenous vascular endothelial growth factor (VEGF) gene was increased by  
3.8-5.2-fold in the C-FABP-transfected cells (pSV-CFABP-R37) and in their  
metastatic sublines (e.g., Met-1) when compared with that in the  
nonmetastatic control transfectant pSV-R37 cells generated by transfection  
of only plasmid DNA. Higher levels of VEGF immunoreactive protein were  
also secreted from the malignant C-FABP-expressing cells. Reverse  
transcription-PCR detected two VEGF transcript isoforms, VEGF164 and  
VEGF188, in both the nonmetastatic control transfectant pSV-R37 cells and  
the malignant metastatic Met-1 cells. Chick chorioallantoic membrane  
assays showed that the conditioned medium of the control pSV-R37 cells  
possessed only very weak angiogenic activity, whereas conditioned media  
from the metastatic C-FABP transfectants and their sublines were strongly  
angiogenic and could be inhibited by antibodies to VEGF. Transfection of  
VEGF164 cDNA in an \*\*\*expression\*\*\* \*\*vector\*\*\* into  
nonmetastatic Rama 37 cells produced a cell clone (R37- \*\*\*VEGF\*\*\* -  
\*\*\*2\*\*\* ) that expressed high levels of VEGF. Inoculation of R37-  
\*\*\*VEGF\*\*\* - \*\*\*2\*\*\* cells into syngeneic Wistar Furth rats produced  
metastases in a significant number (Fisher's exact test, P<0.01) of  
animals (18 of 31 animals), whereas the control, vector alone-transfected  
R37-PSV cells produced no metastases (0 of 30 animals).  
Immunocytochemical methods demonstrated a strong positive staining for  
VEGF and an increased microvessel density in the primary tumors produced  
from PSV- \*\*\*VEGF\*\*\* - \*\*\*2\*\*\* cells in comparison with tumors  
produced from control transfectants. Immunocytochemical staining for  
factor VIII detected a 3.5-fold increase in microvessel density of the  
primary tumors produced by PSV- \*\*\*VEGF\*\*\* - \*\*\*2\*\*\* cells when  
compared with that of the primary tumors developed from the control  
pSV-R37 cells. Therefore, we suggest that overexpression of the C-FABP  
gene in the original transfectants induces metastasis through  
up-regulation of expression of the VEGF gene in this rat Rama 37 model  
system, and thus VEGF may play a crucial role in this particular  
metastatic cascade.

L6 ANSWER 5 OF 8 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS  
INC. on STN

AN 2001:59111 BIOSIS

DN PREV200100059111

TI Active interaction of human A375 melanoma cells with the lymphatics in  
vivo.

AU Papoutsis, Maria; Siemeister, Gerhard; Weindel, Karin; Tomarev, Stanislav  
I.; Kurz, Haymo; Schaechtele, Christoph; Martiny-Baron, Georg; Christ,  
Bodo; Marme, Dieter; Wiltig, Joerg [Reprint author]

CS Lehrstuhl II, Anatomisches Institut der Albert-Ludwigs-Universitaet,  
Albertstrasse 17, 79104, Freiburg im Breisgau, Germany

SO Histochemistry and Cell Biology, (November, 2000) Vol. 114, No. 5, pp.  
373-385. print.  
ISSN: 0948-6143.

DT Article

LA English

ED Entered STN: 24 Jan 2001

Last Updated on STN: 12 Feb 2002

AB We have used the avian chorioallantoic membrane (CAM) to study the  
interaction of tumor cells with the lymphatics in vivo. The vascular  
endothelial growth factor-C ( \*\*\*VEGF\*\*\* - \*\*\*C\*\*\* ) has been shown to  
be lymphangiogenic. We have therefore grown \*\*\*VEGF\*\*\* - \*\*\*C\*\*\*  
-expressing human A375 melanoma cells on the CAM. These tumors induced  
numerous lymphatics at the invasive front, and compressed or destroyed  
VEGF receptor (R)-3-positive lymphatics were observed within the solid  
tumors. The lymphatics in the CAM and in the A375 melanomas could also be  
demonstrated with an antibody against Prox 1, a highly specific marker of  
lymphatic endothelial cells. Proliferation studies revealed a BrdU  
labeling index of 11.6% of the lymphatic endothelial cells in the tumors  
and at their margins. A great number of melanoma cells invaded the  
lymphatics. Such interactions were not observed with \*\*\*VEGF\*\*\* -  
\*\*\*C\*\*\* -negative Malme 3 M melanoma cells. Lymphangiogenesis was  
inhibited to some extent when A375 melanoma cells were transfected with  
cDNA encoding soluble VEGFR-3 (sflt4), and the BrdU labeling index of the  
lymphatics in these tumors was 3.9%. Invasion of lymphatics and growth of  
blood vascular capillaries were not inhibited by the transfection.  
Therefore, tumor-induced lymphangiogenesis seems to be dependent to some  
extent on \*\*\*VEGF\*\*\* - \*\*\*C\*\*\* /flt4 interactions, but invasion of  
lymphatics seems to be a distinct mechanism.

L6 ANSWER 6 OF 8 CAPLUS COPYRIGHT 2004 ACS on STN  
AN 2004:45996 CAPLUS  
TI Construction of eukaryotic \*\*\*expression\*\*\* \*\*\*vector\*\*\* for human vascular endothelial growth factor C gene  
AU Gao, Jie; Liu, Zhiyu; Dong, Ping; Bi, Yushun; Tian, Hua; Li, Guibao; Song, Tao  
CS Department of Anatomy, School of Medicine, Shandong University, Peop. Rep. China  
SO Shandong Daxue Xuebao, Yixueban (2003), 41(2), 151-154  
CODEN: SDXYBZ; ISSN: 1671-7554  
PB Shandong Daxue Xuebao, Yixueban Bianjibu  
DT Journal  
LA Chinese  
AB The eukaryotic \*\*\*expression\*\*\* \*\*\*vector\*\*\* for human vascular endothelial growth factor C ( \*\*\*VEGF\*\*\* - \*\*\*C\*\*\* ) gene was constructed for further study on the role of \*\*\*VEGF\*\*\* - \*\*\*C\*\*\* gene in lymph angiogenesis. According to human \*\*\*VEGF\*\*\* - \*\*\*C\*\*\* cDNA sequence, a pair of specific primers contg. digestion site of EcoR I and BamH I on the 5 end were designed and constructed, then reverse transcription polymerase chain reaction (RT-PCR) was used to clone \*\*\*VEGF\*\*\* - \*\*\*C\*\*\* cDNA from human breast cancer cell MDA-MB-231. After being purified, the product of RT-PCR (1.28 Kb) was ligated into a clone vector pMD18-T. The recombinant plasmid pMD18-T, first propagated in Escherichia coli DH5.alpha., then extd. purified and digested with EcoR I and BamH I, was confirmed to contain full-length \*\*\*VEGF\*\*\* - \*\*\*C\*\*\* cDNA by agarose gel anal. and DNA sequence anal. The resultant EcoR I-BamH I fragment (1.27 Kb) which contained the full-length human \*\*\*VEGF\*\*\* - \*\*\*C\*\*\* cDNA was ligated into eukaryotic \*\*\*expression\*\*\* \*\*\*vector\*\*\* pcDNA3.1(-) digested with EcoR I and BamH I. The pcDNA3.1(-) \*\*\*VEGF\*\*\* - \*\*\*C\*\*\*, digested with EcoR I and BamH I, contained the \*\*\*VEGF\*\*\* - \*\*\*C\*\*\* cDNA sequence identified by agarose gel electrophoresis. The pcDNA3.1(-) \*\*\*VEGF\*\*\* - \*\*\*C\*\*\*, a eukaryotic \*\*\*expression\*\*\* \*\*\*vector\*\*\* for human \*\*\*VEGF\*\*\* - \*\*\*C\*\*\*, was constructed successfully.

L6 ANSWER 7 OF 8 CAPLUS COPYRIGHT 2004 ACS on STN  
AN 2001:265459 CAPLUS  
DN 134:290751  
TI Recombinant single-chain receptor antagonist proteins and their use in treatment of inflammatory disorders  
IN Halkier, Torben; Schambye, Hans Thalsgard; Okkels, Jens Sigurd; Andersen, Kim Vilbourn; Nissen, Torben Laugesgaard; Soni, Bobby; Jeppesen, Claus Bekker; Van Den Hazel, Bart  
PA Maxygen Aps, Den.  
SO PCT Int. Appl., 123 pp.  
CODEN: PIXXD2  
DT Patent  
LA English  
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2001025277	A1	20010412	WO 2000-DK563	20001006
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
EP 1226173	A1	20020731	EP 2000-965860	20001006
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL				
US 2004014948	A1	20040122	US 2003-444691	20030523
PRAI DK 1999-1438	A	19991007		
DK 1999-1855	A	19991223		
DK 2000-1119	A	20000720		
US 1999-160820P	P	19991021		
US 2000-174655P	P	20000106		
US 2000-225723P	P	20000816		
US 2000-684720	B1	20001006		
WO 2000-DK563	W	20001006		

AB The invention relates to a single-chain oligomeric protein antagonist which binds to an extracellular ligand-binding domain of a cellular receptor of a type requiring binding of an oligomeric ligand to two or more receptor subunits to be activated, the protein comprising at least two, typically structurally homologous, receptor-binding sites of which at least one is capable of binding to a ligand-binding domain of the cellular receptor and at least one is incapable of effectively binding to a ligand-binding domain of the cellular receptor, whereby the single-chain oligomeric protein is capable of binding to the receptor, but incapable of activating the receptor; as well as to nucleotide sequences encoding such single-chain oligomeric proteins, expression vectors comprising such a nucleotide sequence, recombinant host cells comprising such a nucleotide sequence or \*\*\*expression\*\*\* \*\*\*vector\*\*\*, methods for producing the nucleotide sequences and proteins, pharmaceutical compns. comprising the single-chain oligomeric protein, and use of the single-chain oligomeric protein for the prodn. of medicaments and in therapy. A preferred single-chain antagonist according to the invention is a TNF-.alpha. antagonist. Thus, a single-chain TNF-.alpha. protein

comprising of 3 human TNF-.alpha. chains connected by linker peptides was produced with Saccharomyces cerevisiae and shown to be an agonist of the TNF-.alpha. receptor. The same TNF-.alpha. trimer contg. Y87R mutations in the first and third copies of TNF-.alpha. was also prepd. This was shown to be a partial TNF-.alpha. agonist and a competitive antagonist of the TNF-.alpha. receptor.

RE.CNT 11 THERE ARE 11 CITED REFERENCES AVAILABLE FOR THIS RECORD

ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 8 OF 8 CAPLUS COPYRIGHT 2004 ACS on STN  
AN 1998:46307 CAPLUS  
DN 128:152421  
TI Down-regulation of vascular endothelial growth factor in a human colon carcinoma cell line transfected with an antisense \*\*\*expression\*\*\* \*\*\*vector\*\*\* specific for c-src  
AU Ellis, Lee M.; Staley, Charles A.; Liu, Wenbiao; Fleming, R. Y. Declan; Parikh, Nila U.; Bucana, Corazon D.; Gallick, Gary E.  
CS Departments Surgical Oncology, Cell Biology, Univ. Texas M.D. Anderson Cancer Center, Houston, TX, 77030, USA  
SO Journal of Biological Chemistry (1998), 273(2), 1052-1057  
CODEN: JBCHA3; ISSN: 0021-9258  
PB American Society for Biochemistry and Molecular Biology  
DT Journal  
LA English  
AB Vascular endothelial growth factor (VEGF) is implicated in the angiogenesis of human colon cancer. Recent evidence suggests that factors that regulate VEGF expression may partially depend on c-src-mediated signal transduction pathways. The tyrosine kinase activity of Src is activated in most colon tumors and cell lines. The authors established stable subclones of the human colon adenocarcinoma cell line HT29 in which Src expression and activity are decreased specifically as a result of a transfected antisense \*\*\*expression\*\*\* \*\*\*vector\*\*\*. This study detd. whether VEGF expression is decreased in these cell lines and whether the smaller size and reduced growth rate of antisense vector-transfected cell lines in vivo might result, in part, from reduced vascularization of tumors. Northern blot anal. of these cell lines revealed that VEGF mRNA expression was decreased in proportion to the decrease in Src kinase activity. Under hypoxic conditions, cells with decreased Src activity had a <2-fold increase in VEGF expression, whereas parental cells had a >50-fold increase. VEGF protein in the supernatants of cells was also reduced in antisense transfectants compared with that from parental cells. In nude mice, s.c. tumors from antisense transfectants showed a significant redn. in vascularity. Apparently, Src activity regulates the expression of VEGF in colon tumor cells.

RE.CNT 38 THERE ARE 38 CITED REFERENCES AVAILABLE FOR THIS RECORD

ALL CITATIONS AVAILABLE IN THE RE FORMAT

=>

---Logging off of STN---

=>

Executing the logoff script...

=> LOG Y

COST IN U.S. DOLLARS	SINCE FILE	TOTAL
	ENTRY	SESSION
FULL ESTIMATED COST	43.53	43.74

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